small bowel contents (for example, bile acids, cholesterol) to large bowel mucosa; such altered gastrointestinal metabolism might predispose to anorectal carcinoma just as dietary changes are thought to predispose to colorectal carcinoma. The role of microflora and bile acids in producing carcinogens or co-carcinogens from substrate in the gut has yet to be elucidated. Finally, the fatty acids and bile acids present in the severe diarrhea could be irritating enough to large bowel mucosa to serve as preliminary antigenic stimulants to a chronic inflammatory response which subsequently becomes dysplastic.

Patients who have had a jejunoileal bypass operation for obesity commonly suffer anorectal complaints. This discovery of anorectal carcinoma in one such patient underscores the importance of an adequate physical examination and workup. Although the occurrence of anal carcinoma in this patient may have been unrelated to the jejunoileal bypass procedure, several sequelae of jejunoileal bypass may predispose to colorectal and anal malignancy.

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Familial Hiatal Hernia

TO THE EDITOR: Congenital diaphragmatic defects have been reported to be familial.[1,2] On the other hand, hiatal hernia has generally been regarded as a sporadic nongenetic malformation with practically no risk of recurrence in subsequent pregnancies. A family with six affected persons is presented here. To my knowledge, this is the first report of familial occurrence of hiatal hernias.

A 13-month-old child had a long history of persistent regurgitation dating from the newborn period. She had four to five regurgitant episodes per day. The regurgitation was worse when she was in a horizontal position. She was a product of an uncomplicated pregnancy, full-term normal spontaneous delivery. Birth weight was 3,180 grams. Past health was unremarkable. Paternal great-grandfather, grandmother, grandaunt as well as maternal great-grandmother, grandmother and grandaunt had sliding hiatal hernias confirmed radiologically. Paternal grandaunt, maternal great-grandmother and maternal grandaunt had operative repair of hernias done. The rest of the affected family members were treated with antacids. There is no consanguinity. Physical examinations of the patient showed no abnormalities. Radiographic barium contrast studies of the upper gastrointestinal tract showed pronounced gastroesophageal reflux to the level of the cricopharyngeus.

Hiatal hernia has a multifactorial mode of inheritance although the specific cause is unknown. Most reviews fail to mention the possibility of familial occurrence. The above cases suggest a hereditary factor in the genesis of hiatal hernia. As with other patients with hiatal hernias, the index patient has gross gastroesophageal reflux. Whether this will lead to a sliding hiatal hernia at a later date, for example, through an esophageal stricture formation, is unknown. With such a strong family history, this possibility cannot be excluded.

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Designing New Devices for Measuring Blood Pressure

TO THE EDITOR: In their excellent clinical article on effect of stethoscope pressure on blood pressure measurement,[1] Londe and Klitzner did not examine newer devices for blood-pressure monitoring such as digital readout home blood pressure cuffs and Doppler cuffs that do not use a stethoscope but nevertheless do apply a sensor to the skin under pressure. Would these devices also result in artifactual decrease in diastolic blood pressure readings? Judging from the data presented, one would assume that if these instruments were designed so that less than 10 mm of mercury pressure would always be applied by the sensor, they would appear to have a potential advantage in uniformity over fallible human hands—a considerable advantage in blood pressure screening projects of all kinds. One would hope that manufacturers of these new high-tech wonders will take Londe and Klitzner's work into consideration in future design and manufacturing processes.

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Private Sector Financing for Medical Education

TO THE EDITOR: Mr Barna astutely noted in his July letter[1] that there seems to be a "physician glut and a coexisting and paradoxical physician shortage." I agree that the production of more physicians will "filter down" fewer doctors for Tulelake than for Beverly Hills. However, few physicians in training base their future practice locale or their specialty choice on the size of their educational debt. Most students are too inundated with scientific esoterica to be concerned with such economic "facts." Our economic naivete is manifest in that there are much better ways for intelligent people to make money than practicing medicine.

Mr Barna also pointed out that the California AHEC program results section did not designate actual numbers of physicians settling in underserved areas. Most physician manpower decisions are based on this type of physician census and physician/population ratios which are inaccurate representations of physician "supply." Ratios exclude such issues as inner city problems of accessibility to professional medical care. A census implies equal productivity for each individual

physician. Maldistribution is a complex issue which is better defined by each community's assessment of its own supply rather than relying on census figures or similar determinations.

As a participant in Ohio's Preferred Placement Program (PPP), I support the concept to finance medical education in return for future services in both rural and urban underserved areas. PPP has developed a financial instrument ($1.5 million) whereby local communities raise private sector monies to finance a student's medical education in return for practice obligations. The physician glut predicted by the Graduate Medical Education National Advisory Committee (GMENAC) encourages the dwindling of government subsidies for medical education. Whether this will filter down to a surplus of physician services for urban and rural underserved communities has yet to be determined. Organized medicine should support and lobby for programs that organize private sector monies for students both to insure equal socioeconomic access to medical education and to help curb maldistribution.

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Oral Contraceptives and Venous Thrombosis

TO THE EDITOR: In his article in the journal[1] on the systemic effects of oral contraceptives, Dr Thomas Kelly states, "In contrast to the controversial relationship between oral contraceptives and arterial occlusive disease, the data regarding the effect of their use on venous thrombosis are quite straightforward." Indeed, they are straightforward; but the scientific interpretation is quite different from that given by Dr Kelly. In a review published in 1973,[2] I pointed out that the association between ingestion of oral contraceptives and venous thrombosis found in retrospective studies could be accounted for by the wide publicity given previously in the medical and lay press of a possible risk of venous thrombosis in oral contraceptive users. As a result, any woman taking the pill and presenting with leg or chest pains would have been carefully examined for evidence of venous thrombosis or pulmonary embolism and many would have been admitted early to a hospital for further studies, such as venography. On the other hand, similar symptoms in a woman not taking the pill might have been dismissed by the patient or her physician as being merely due to leg cramps.

This hypothesis of a greater index of suspicion of venous thrombosis in women using the pill than in nonusers was subsequently supported by the studies of Barnes and coworkers in 1977.[3] They found that the incidence of venous thrombosis proved by Doppler examination in women taking oral contraceptives in whom venous disease was suspected was only 16.7% which was about half the incidence documented by Doppler in women not taking the pill. Furthermore, most prospective studies have not shown an increased incidence of venous thrombosis in women taking the pill. Indeed Dr Ramcharan, the principal investigator in the very large and well-controlled Walnut Creek study, concluded at the end of the study that oral contraceptives as a cause of thromboembolism had not been proved since the effect of diagnostic bias was a strong possibility. Moreover, a cause-and-effect relationship cannot be postulated merely on the basis of a statistical association.

That oral contraceptives induce an increase in certain clotting factors and a slight decrease of antithrombin III is not in dispute. However, no worker with any recent experience in the field of blood coagulation would any more deduce from these facts that these changes tip the hemostatic balance towards thrombosis than they would postulate that a lengthening of the clotting time necessarily indicates hypocoagulability. The facts are now very clear. But although there is no proof that contraceptives predispose towards venous thrombosis, it remains the general belief that a cause-and-effect relationship has been established and this is repeated from one textbook to another without any critical evaluation of the data on which the original claim was based.

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Dr Kelly Replies

TO THE EDITOR: Dr Hougie suggests that the preconceived impressions of epidemiologists who studied oral contraceptive effects caused them to intensively investigate symptomatic users of oral contraceptives and thus find a spurious association of those agents and venous thrombosis. The designers of early British studies of oral contraceptives were well aware that bias could influence retrospective, case-control studies, and they thoroughly discussed in their reports why bias was unlikely to explain the results they had obtained.[1] In particular, they found that diagnostic studies for venous thromboembolic disease were not employed more frequently in patients using oral contraceptives than in control subjects.[2]

To support his argument that physicians more readily study for thromboembolic disease women receiving oral contraceptives, Dr Hougie references a report in which women referred for Doppler evaluation of leg veins were only half as likely to have confirmation of thrombosis if they were taking oral contraceptives than if they were not. That result, however, is not a universal finding as others have reported observing no difference in the percent of venograms positive for deep venous thrombosis when young women with clinically suspected disease were divided according to oral contraceptive use. Tibbutt found that about twice as many oral contraceptive users were referred for study and at least twice as many had documented thromboses.[3]